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DISEASES of the NERVOUS SYSTEM

A PRACTICAL JOURNAL ON PSYCHIATRY AND NEUROLOGY

Mestinon in Myasthenia Gravis

(Preliminary Report)

J. E. TETHER, M.D.
Indianapolis, Indiana

Although neostigmine is currently the drug of choice in the treatment of myasthenia gravis, it has adverse characteristics which make it far from the ideal drug for the alleviation of the symptoms of this disease. Some failings of neostigmine are: 1) the intensity and frequency of untoward reactions, particularly gastrointestinal; 2) the relatively short duration of action which makes it difficult for the myasthenic to provide for a residual effect in the early morning hours; and 3) the intensity of action of neostigmine is such that the myasthenic is continuously subject to peaks and valleys rather than experiencing a smooth, sustained, plane effect. Recent experience with a new cholinergic agent which overcomes some of the disadvantages of neostigmine has been sufficiently encouraging to warrant the preparation of this preliminary report.

A number of reports have appeared in the European literature describing the properties of this analogue of neostigmine.[1-10] The material has been referred to by one or more of the following designations: Nu 1317, Nu 5130, prostigmin-5130, Ro 2-1317, Ro 1-5130 and Pyridostigmin. The bromide salt of the drug was made available to us under the name "Mestinon."

A report of the administration of "Nu 1317" to a myasthenic patient was made by Millikan and Eaton.[11] Seibert[12] has reported his favorable experience with 23 patients and Bauer and Schmid[13] have also published their findings. In late 1947 we used Mestinon on 6 myasthenics. The results were equivocal with the exception that there were fewer reactions than with neostigmine. In retrospect, our findings at that time were probably due to the low dosage used (30 mg).

Procedure:

Sixty-eight courses of Mestinon were given to 56 myasthenics. These patients were of all types, from the mildest to the most severe. Some had been maintained on a fairly steady dosage schedule of neostigmine (oral) for many years without remissions or relapses. Others, predominantly females, had experienced real difficulty in regulating their neostigmine requirements, either because of frequent partial remissions and relapses, or due to either unusually rapid or delayed gastrointestinal absorption.

In our study there were 13 males and 43 females. The average age was 40 years, the range 12 to 72 years. The average duration of the disease was 8.3 years, range 1 to 41 years. Mestinon bromide was administered orally in tablets containing 50 to 60 mg each. During the early course of the study, Mestinon gradually replaced neostigmine on the basis of 50 mg Mestinon bromide to 15 mg neostigmine bromide. As further experience with the drug was obtained, patients were changed directly from neostigmine to Mestinon without a transition period except in the most severe myasthenics.

From Department of Neurology, Indiana University Medical Center.

TABLE I

Summary of Treatment with Neostigmine and Mestinon Bromide

No.	Case	Age (yrs)	Sex	Duration Disease (yrs)	NEOSTIGMINE			MESTINON BROMIDE			
					Daily Dose (mg.)	M.G. Symptoms with Therapy	Reactions	Daily Dose (mg.)	Duration Treatment (weeks)	M.G. Symptoms with Therapy	Reactions
1	E.A.	68	M	1	960	1.5	1	3200	1	2.5	0
2	V.A.	24	M	13	330	1.0	3	600	1	2.5	0
	ditto							2400	2	0.5	0
3	M.B.	46	F	10	75	1.5	4	200	4	0.5	2
4	L.B.	47	F	1	30	0.5	1	75	1	0	0
5	P.B.	46	F	3	75	1.0	4	150	4	0.5	0
6	L.B.	47	F	2	525	2.0	4	1750	3	3.0	0
7	F.B.	29	F	5	30	1.5	1	75	4	0.5	0
8	J.C.	41	M	1	180	1.0	0	600	2	3.5	0
9	M.C.	35	F	13	255	1.5	4	240	1	0	0
10	E.C.	48	F	18	480	2.5	2	1300	3	1.5	1
11	T.C.	38	F	1	60	1.5	4	150	5	0.5	1
12	J.C.	39	M	20	60	0.5	1	150	1	0	1
13	M.D.	30	F	2	480	1.5	1	1000	1	0.5	0
14	S.D.	54	M	5	60	1.5	4	300	2	0.5	0
15	Z.D.	37	M	10	135	1.0	3	225	13	0.5	1
16	V.F.	53	F	40	225	0.5	1	600	1	2.0	1
17	W.F.	40	F	5	225	1.5	3	175	1	0	0
18	E.F.	50	F	7	1920	3.5	0	4800	1	3.5	0
	ditto*				750	3.0	0	2400	4	2.0	0
19	J.G.	29	F	20	120	0.5	3	300	2	0.5	1
	ditto							350	3	0	1
20	B.H.	27	F	20	135	1.0	4	175	4	0.5	0
21	T.H.	41	F	2	300	2.0	2	400	2	1.5	0
22	J.H.	36	F	2	90	1.0	2	200	1	0.5	0
23	M.K.	32	F	3	90	0.5	3	150	3	0	0
24	V.K.	33	F	2	360	2.5	4	1200	7	2.5	0
25	W.L.	27	F	4	255	1.0	4	350	5	0.5	0
	ditto							1000	3	2.0	2
26	V.L.	50	F	10	60	0.5	4	175	9	0	0
27	L.L.	39	F	10	810	1.5	2	1350	4	0.5	0
28	P.L.	22	F	10	300	2.0	4	750	8	1.5	0
29	F.M.	45	F	3	225	2.5	4	500	2	2.5	0
30	V.Mc.*	40	M	5	540	2.5	3	250	3	1.5	1
31	S.M.	12	M	12	180	0.5	0	300	3	0.5	0
32	P.M.	27	F	2	90	1.0	3	150	2	1.5	1
	ditto							225	8	0	0
33	M.M.	36	F	1	45	0.5	4	200	2	0	2
	ditto							75	4	0	0
34	R.N.	34	F	2	150	1.5	4	500	8	0.5	0
35	T.P.	57	F	7	60	1.5	4	150	4	0	0
36	C.P.	40	F	4	375	2.0	2	900	2	2.0	1
37	A.P.	40	F	13	525	2.0	0	1000	3	1.0	0
	ditto							2000	7	0	0
38	C.P.	41	F	8	105	1.0	3	75	12	0	0
	ditto							100	6	0	0
39	W.P.	27	M	1	1680	3.0	2	3299	12	2.0	0
40	A.R.	40	F	10	450	2.0	4	1000	3	1.5	0
41	D.S.	41	F	41	75	1.0	2	150	4	0.5	0
42	M.S.	28	F	2	2700	3.5	3	4800	1	3.0	0
43	C.S.	38	F	10	420	2.0	0	2400	3	2.5	0
	ditto				900	1.5	1	2400	4	2.5	0
44	D.S.	36	F	6	120	1.0	4	275	8	0.5	0
45	K.S.	56	F	2	230	1.5	2	459	3	0	2
	ditto				195	2.5	4	500	3	1.5	0
46	G.S.	33	F	18	45	1.5	1	150	3	2.5	2
	ditto							100	1	1.0	0
47	L.S.	47	F	2	360	3.0	2	1200	1	2.5	1
48	N.T.	57	F	3	270	1.5	4	900	6	2.0	1
	ditto				300	2.5	4	750	11	1.5	0
49	L.T.*	45	M	10	75	1.0	4	175	5	0.5	0
50	R.T.	32	M	4	120	2.5	2	250	4	0.5	2
51	I.T.	72	F	5	105	1.5	1	300	1	2.5	3
52	A.W.	45	F	10	90	2.5	0	400	4	0.5	0
53	H.W.	39	M	8	150	1.0	2	500	3	0.5	0
54	V.W.	44	F	3	450	2.5	4	600	9	1.5	0
55	A.W.	35	F	5	60	1.5	2	200	7	0.5	0
56	R.Z.	55	M	30	225	1.5	3	200	8	0.5	0
Average		40		8.3	345	1.6	2.5	796	4.1	1.1	0.4

*In partial remission.

Results:

The results of this preliminary study are summarized in Table I. A numerical code indicating roughly the severity of myasthenic symptoms while under therapy, and the degree of reactions to the drugs was established as follows: 0—no symptoms; 1—slight; 2—mild; 3—moderate; 4—severe. Dosage figures in the table indicate the average during each course of treatment. In some cases, remissions may account for relatively lower dosage of one drug as compared with the other.

The daily dose of neostigmine bromide ranged from 30 to 2700 mg. The average was 345 mg/day. The average severity of myasthenic symptoms under neostigmine therapy was 1.6 (slight to mild). The average degree of neostigmine reactions was 2.5 (mild to moderate).

The daily dose of Mestinon bromide varied from 75 to 4800 mg. The average was 796 mg/day. The duration of treatment with Mestinon ranged from 1 to 13 weeks, the average being 4.1 weeks. The average severity of myasthenic symptoms under Mestinon therapy was 1.1 (slight). The average degree of reactions to the drug was 0.4 (none to slight). These data indicate that both neostigmine and Mestinon provided relatively equal control of myasthenic symptoms (1.6 vs. 1.1). These averages, however, do not adequately convey the sustained and smoother course under Mestinon.

In several instances, when Mestinon was thought to be inferior to neostigmine on the first trial, a second course on heavier dosage gave opposite results. In one case, G. S. (No. 46), a slight reduction of dosage on the second course gave superior results. Mild myasthenics, who had been taking small doses of neostigmine irregularly for intermittent symptoms, objected to the slowed onset of action of Mestinon as compared with neostigmine.

The lesser reaction potential of Mestinon is shown in two ways. First, the average severity of reactions differ, 2.5 for neostigmine, 0.4 for Mestinon. Second, in only 7 instances were there no reactions to neostigmine, whereas this occurred 49 times with Mestinon. A severe neostigmine reaction (Grade 4), consists of severe muscle fasciculation, with increased muscular weakness, and signs of pronounced parasympathetic overactivity, including flushing, sweating, salivation, nausea, vomiting, intestinal cramping and diarrhea. These usually occur when a patient fails to reduce his dosage in the face of a sudden remission. The most severe reactions thus far experienced by our patients taking Mestinon have consisted of mild fasciculation, with weakness; and mild flushing, intestinal cramping, and diarrhea. These Mestinon reactions have rarely been of sufficient severity to require atropine or belladonna, and then only as a precautionary measure.

As with neostigmine, no unusual toxicity, as indicated by changes in blood or urine, has thus far been encountered in any of the patients taking Mestinon, even with extremely heavy dosage.

A comparison of neostigmine and Mestinon is illustrated by the experience of patient L.S. (No. 47), who, on a daily dosage of 360 mg. of neostigmine, went into crisis on December 15, 1953. A tracheotomy was done and she was placed in a respirator. Neostigmine was stopped for four days, as it was felt that she was in cholinergic crisis. It was then cautiously resumed, parenterally, and gradually increased to 1 mg. every two hours. When this dosage was reached, voluntary respiration returned, and the respirator was discontinued. However, she still could not swallow well, and excess secretions were a constant problem, requiring atropine, especially at the height of the neostigmine effect. She was placed on oral Mestinon on January 10, 1954 when her dosage reached 1200 mg. daily (150 mg. every 3 hours), her ability to swallow and her general strength rapidly improved, and secretions diminished greatly, so that her tracheotomy tube could be removed. On January 28, 1954, excess secretions and weakness reappeared, but diminished when her Mestinon dosage was reduced to 60 mg. every 3 hours.

Discussion:

Until 1934, when Walker[14] reported the remarkable therapeutic effect of neostigmine on myasthenia gravis, and 1935, when Viets and Schwab[15] described their neostigmine diagnostic test, myasthenia was thought to be an extremely rare medical curiosity. Since

that time, an ever increasing number of diagnoses has been made, especially in centers where any real interest in the disease exists. This trend has been given further impetus by the use of provocative tests with quinine,[16] and curare,[17] and by intravenous neostigmine,[18] and Tensilon tests.[19]

In the past twelve years, the author has seen 456 cases in whom easy fatigability of voluntary musculature, local or general, has either been diagnostically aggravated by one-tenth of a minimal curarizing dose of d-Tubocurarine, or relieved by intravenous neostigmine, or, in most cases, both. They have thereafter shown positive responses to neostigmine bromide taken by mouth. They have thus satisfied the diagnostic criteria for myasthenia gravis. Many of these cases have classically severe myasthenia gravis. Many others have typical myasthenic symptoms in milder degree. This situation seems analogous to that seen in such conditions as diabetes and hyperthyroidism, in which there are many mild cases for every severe one.

Tolerance for and requirements of neostigmine are, in most cases, proportional to the degree of myasthenia. We have seen dosages rise to 250 tablets (15 mg. each) of neostigmine bromide daily in unusually severe myasthenia, without intolerance. On the other hand, some patients cannot completely control their symptoms on the maximum tolerated dose. Others, apparently because of rapid gastrointestinal absorption, experience overdosage reactions shortly after taking the drug, followed by a short period of increased strength, and then a rapid reappearance of weakness. Such patients are best controlled by giving milk with frequent small doses of neostigmine. Food seems to slow up and smooth out absorption (which may mean that hyperacidity is one of the causes for over-absorption of neostigmine). In spite of these measures, an occasional patient must resort to parenteral medication for its more predictable action.

The course of myasthenia is characteristically marked by remissions and relapses. Thus, a set dosage schedule is seldom satisfactory. Therefore, it has been the practice of this author to place all patients on demand dosage as soon as possible. It is remarkable how quickly most of them learn to judge their proper dosage requirements. Nevertheless, some patients are subject to such rapid fluctuations that they are almost always either underdosed or having mild neostigmine reactions.

Thus, it becomes apparent that a successor to neostigmine should possess the following characteristics:

1) It should be more slowly and evenly absorbed from the gastrointestinal tract.

2) It should have a longer, steadier curve of activity within the body.

3) It should give milder side-effects, or none at all.

Mestinon bromide, on the basis of this preliminary report, seems to possess these characteristics. The patients frequently described the course of action of Mestinon as "smooth," "sustained," "stronger in the morning." All of these are indicative of either a more even rate of absorption, a more prolonged duration of activity, or both. In the patients estimate, Mestinon was poorer than neostigmine in 12 courses, equal in 5, questionable in 5 and superior to neostigmine in 46.

Summary and Conclusions:

1. Mestinon bromide was administered to 56 myasthenics. The average dose was 796 mg/day and the average duration of therapy 4.1 weeks.

2. Mestinon is less desirable than neostigmine because it has a slower onset of action which is a disadvantage in patients who frequently experience sudden severe relapses.

3. Mestinon has the following advantages over neostigmine as oral therapy for the average myasthenic:

(a) It is more slowly and more evenly absorbed, or has a more prolonged duration of activity in the body, or both. Thus it gives smoother, more sustained control of symptoms.

(b) Untoward reactions are less frequent and less intense.

4. No toxicity was noted in any of the patients treated with Mestinon in the series.

Acknowledgment: The author wishes to express his deep appreciation to Dr. M. J. Schiffrin of Hoffmann-La Roche, Inc., who gave valuable advice and assistance, and to his company, for furnishing the Mestinon used in this study.

Addendum: Since this paper was submitted for publication, about 100 additional myasthenics have been given trial courses of Mestinon. Results have been similar to those reported here, and will be included in a later report.

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The Electroencephalogram in Blood Diseases:

(Abnormalities Observed in Two Patients)

SAMUEL M. TARNOWER, M.D., HENRY J. WHEELWRIGHT, M.D.
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Marked elevation of the platelet count may be seen in certain blood dyscrasias such as polycythemia vera, the leukemias, and Hodgkin's disease. Neurologic symptoms commonly occur in these diseases and such nervous system involvement has been described by many observers. "Polycythemia vera and the nervous system" was reported by Johnson and Chalgren in 1951.[1] They made an exhaustive review of the literature and added their personal observations in five patients. In summary, they estimated that symptoms related to the nervous system are present in at least three-fourths of all cases of polycythemia vera. Further, they considered that in a third of all cases, neurologic symptoms are the most important complaint. The most common symptoms are non-specific and include headache, vertigo, fatigue, and weakness. In 1935 Schwab and Weiss[2] reviewed all previous literature regarding the involvement of the nervous system in leukemia. They showed that such neurologic complications were much more common than was realized at that time. Wintrobe's textbook of hematology[3] gives adequate mention of such nervous system lesions. He states that "any part of the nervous system may be involved in leukemia and the lesions may take the form of hemorrhage, thrombosis, infiltration with leukemic cells, or tumor-like growths."

When one considers that neurologic symptoms are known to be associated with some blood diseases, it is surprising that there is little mention in the literature of electroencephalographic studies in these clinical states. Several textbooks have been published in the field of electroencephalography but only one of these mentions such studies in blood diseases. The authors (Strauss, Ostow, and Greenstein[4]) state that three patients with blood dyscrasias (aplastic anemia, secondary anemia, and polycythemia) showed normal records. In a patient with pernicious anemia they found a mildly abnormal record. A recent electroencephalographic journal[5] makes first mention of an extensive study of electroencephalograms in patients with pernicious anemia and subacute combined system disease. This report is a summary of society proceedings of The EEG Society in London on May 2, 1953. The authors found a significant percentage of electroencephalographic abnormalities in the 130 patients studied. They felt that their findings revealed a definite correlation between clinical and electroencephalographic improvement. One of us (S.M.T.) reported our findings in abstract form to the Third International Congress of Electroencephalography and Clinical Neurophysiology which met in Cambridge, Massachusetts, from August 17-22, 1953.[6]

At this time we are reporting our findings in two patients with blood disease. It will be noted that we have only one electroencephalographic record of the first patient. The diagnosis was leukemia of the myelogenous type with megakaryocytosis and thrombocytosis. The significance of her electroencephalogram was realized only after the more impressive abnormalities were found in the second patient some time later. The latter patient has polycythemia vera and the usual associated thrombocytosis. We have obtained many records in her case but are presenting only the most important ones at this time.

Case 1. L.Y., a 57 year old white widow was admitted to the care of Dr. Herbert Glodt at the Pittsfield General Hospital on February 13, 1952. She complained of headaches which had persisted for two months with increasing severity. The head

From the Medical Department and the Electroencephalographic Laboratory of the Pittsfield General Hospital.

pain was throbbing in character, was greater on the left side and was aggravated when the patient was in the upright position. About two weeks before admission she noticed some difficulty in pronouncing words. Physical examination was not remarkable except for atrophy and weakness of the left tongue. Skull x-rays were normal but an electroencephalogram was diffusely abnormal, the slow activity being more prominent on the left side. Blood studies on this admission were as follows: Hgb. 11.4 gm.%, red blood count 3,590,000 per cu. mm., white blood count 11,000 per cu. mm., 68% neutrophils, 30% lymphocytes, 2% monocytes. A smear showed achromia with variation in size and shape of the red cells. The patient was transferred to the Albany Hospital for further neurologic investigation. At the latter hospital, blood examinations revealed a thrombocytosis associated with an aleukemic leukemia of the granulocytic type. Subsequently this patient was re-admitted several times to the Pittsfield General Hospital for treatment. Such admissions were occasioned by increased headache, vomiting, and vertigo. All follow-up blood findings were considered to be characteristic of leukemia. A typical blood study was reported on March 3, 1952: Hgb. 9.5 gm.%, red blood cells 3,500,000 per cu. mm., white blood cells 14,980 per cu. mm., neutrophils 60%, lymphocytes 20%, monocytes 4%, eosinophils 6%, basophils 4%, myelocytes 6%, and a platelet count of 1,792,000 per cu. mm. She developed mental symptoms which were typical of organic brain involvement. She died on June 28, 1952. An autopsy was performed and the findings were reported by the pathologist, Dr. Helen M. Scoville, as follows: "L.Y., Anatomical Diagnosis: Moderate splenomegaly; subpleural fibrosis and congestion of the posterior borders of the lungs; acute infarct of the right kidney; focal leukemoid infiltration of the meninges; leukemoid cuffiing of some of the cerebral vessels; marked engorgement of the pituitary gland. Note: The post mortem examination showed evidence of leukemia primarily in the spleen and in the meninges and brain. There was nodular infiltration of the meninges which was striking, grossly and microscopically. This is not a common finding but is reported to occur in approximately 7% of the patients who die of leukemia. While there was no actual necrosis of the brain there was marked cuffiing of part of the smaller blood vessels with leukemic cells and this would seem to be a reasonable explanation of many of the clinical symptoms. Many of the small blood vessels in the meninges and throughout the body showed an uneven clumping of the blood cells within the lumena, but no typical recognizable ante mortem clot. This seems somewhat unusual in view of the very high platelet count that was found during life; one platelet count sometime before death is recorded as high as a million platelets per cu. mm. The total count seems to have fluctuated somewhat but while no platelet count was done just before death, it was noted that there were many large clumps of platelets quite obvious in the preparations used for doing routine blood counts."

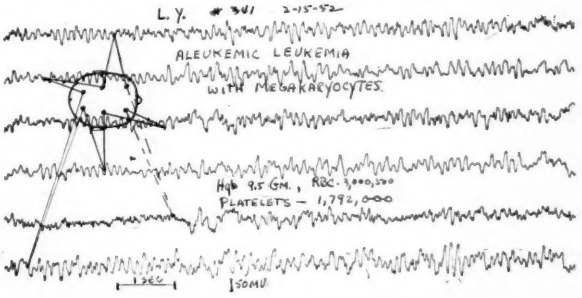

Figure 1. Patient L.Y. Aleukemic leukemia with thrombocytosis. The record is abnormal showing a moderate amount of 5 to 7 per second activity and some 2 to 3 second activity.

Case 2. E.Mc., a 62 year old housewife, was first seen by us in March of 1950 at her home because of severe headaches present for the previous ten years. The headaches tended to start after hard work without prodromata, were usually right sided and tended to spread to the left. The headaches were pounding in character. The headaches had recently been associated with nausea and came every one to two months. They had not been helped by Cafergon. Physical examination was negative save for a blood pressure of 160/80 mm. She was next seen in December of 1951 because of severe nosebleeds. A blood pressure at that time was 190/100 mm. and she still complained of headaches though she stated that they were relieved by aspirin and an icebag. On September 1, 1952 she developed a generalized headache and dizziness. On awakening from a nap she was trembling and confused. On examination her blood pressure was 220/120 mm. There were increased deep reflexes of the left arm and leg. Hospitalization was suggested but refused. Within a month's time she was much improved but still showed increased deep reflexes of her extremities on the left. In April of 1953 there was some recurrence of headaches and considerable confusion. She complained of shooting lights before her eyes and increasingly severe headaches which were pounding in character. She was unable to be out of bed for any length of time. Physical examination showed a blood pressure of 180/100 mm. and a normal temperature. Her spleen for the first time was felt two fingers' breadth below the left costal margin. There were no abnormal neurological findings save for an equivocal left facial paralysis. The patient was hospitalized for investigation. An initial blood count showed the following: Hgb. 21 gm.%; white blood cells 9,900 per cu. mm. with neutrophils 74%, lymphocytes 17%, monocytes 5%, and eosinophils 4%. The red blood cells showed hyperchromia. The hematocrit was 66% with a platelet count of 721,000 per cu. mm. Spinal fluid studies and an x-ray of the skull were normal but an electroencephalogram showed a frankly abnormal pattern. It was felt that her signs and symptoms not only present but past were on the basis of a polycythemia vera. Phlebotomy was attempted but careful search failed to reveal any accessible veins; in con-

sequence leechs were applied with a reduction in her red blood count and considerable clinical improvement. Subsequently she was given Phenylhydrazine followed by Triethylene Melamine. By the time of her discharge on May 23, 1953 she was markedly improved. Her headache was gone and her weakness was diminished. At the time of discharge, the complete blood count was as follows: Hgb. 12.8 gm.%, red blood cells 3,800,000 per cu. mm., white blood cells 13,000 per cu. mm. and a platelet count of 800,000 per cu. mm. When seen on August 3, 1953 she felt well and was free of headaches. There were no neurological abnormalities. Her blood pressure was 155/85 mm. Hg. On repeated subsequent examinations she complained on some weakness and occasional mild headache. Repeated electroencephalograms showed disappearance of the abnormal electrical activity.

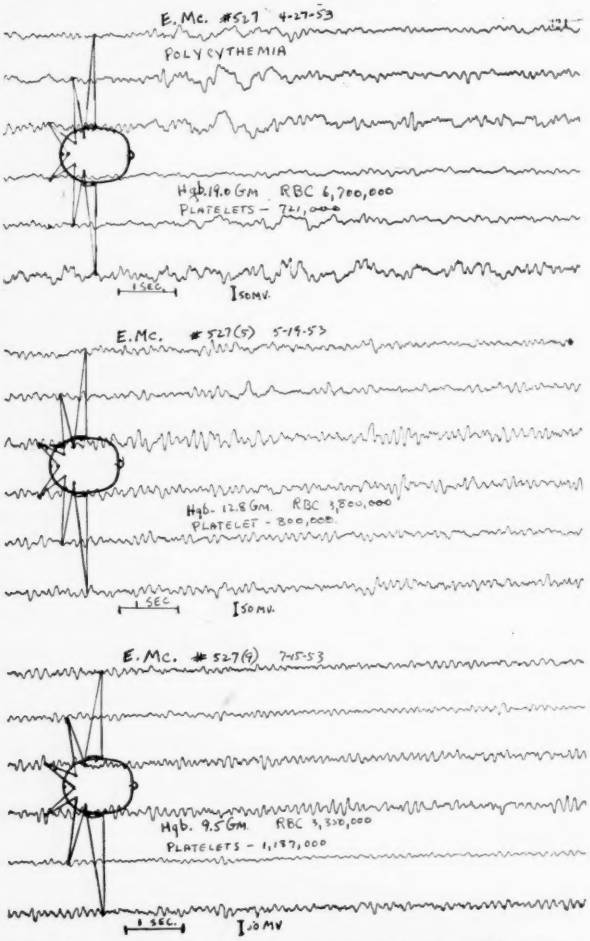

Figure 2. Patient E.Mc. Polycythemia. This record is abnormal. There is diffusely distributed slow activity down to 1½ cycles per second.

Figure 3. Patient E.Mc. After a 22 day interval the EEG shows definite improvement. At this time the red cells and hemoglobin were normal but the platelets were still increased.

Figure 4. Patient E.Mc. This record was taken 79 days after the initial EEG of April 27, 1953. The record shows marked improvement and is considered to be normal. At this time the platelet count had reached its highest levels while the red cells were moderately depressed.

Discussion

Thrombocytosis was found in the two patients described in this report. We felt that this common factor might have special significance since the patients had almost identical presenting complaints of headache. However, our later findings suggested that other factors might be as important as the increased platelets. It can be seen that the patient with polycythemia improved clinically and electroencephalographically when the increased number of red cells returned to normal. The improvement took place while the platelet count remained high. This suggests that not only thrombocytosis but also other changes, such as increased red blood cells or increased white blood cells, may cause neurologic lesions and the resultant electroencephalographic abnormalities. Such lesions may be due to circulatory stasis with tissue anoxia and to disturbances of function secondary to vascular lesions. In leukemia, infiltrations and leukemic tumors may occur. An interesting observation in our patients was the marked contrast between the relatively normal neurologic findings and the abnormal electroencephalographic records. This was best seen in the second patient, E. Mc. She complained of severe headache but objective neurologic examination revealed no definite abnormalities. Her electroencephalograms, however, showed prominent abnormal slow waves.

The abnormal electroencephalograms in these two patients with blood disease represent a laboratory confirmation of previously described neurologic involvement. We wondered if patients with blood diseases had been studied previously by others and had been found normal. We are now of the opinion that electroencephalographic studies are rarely done in such patients since the primary diagnosis is usually established early by routine studies. The neurologic symptoms which occur in patients with blood diseases are probably accepted as a known type of complication and further investigation may seem unnecessary to the clinician. The electroencephalograms of our patients were requested because of the prominent complaint of headache, before the hematologic diagnosis was established. The two cases reported at this time demonstrate that

the electroencephalogram may be grossly abnormal in patients with blood disease.

Summary

Thrombocytosis and other altered blood states in polycythemia vera and leukemia may cause a great variety of neurologic complications. Evidence of such nervous system involvement was found in the electroencephalograms of the two patients included in this report. It is felt that the more widespread use of the electroencephalogram in such diseases will reveal significant clinical and hematologic correlations. The electroencephalographic phenomena described here have not been previously reported by others.

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A Rare Intracranial Lesion

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This case report is presented in order to show the many difficulties encountered in correctly establishing the neurological diagnosis and to show the course of the condition from a relatively early stage to its terminus.

E. C., male, age 45, was admitted to a Diagnostic Center of the Veterans Administration on October 2, 1942, for a period of examination and observation to establish a definite diagnosis. The medical history revealed no illness of note, including venereal disease, prior to January 31, 1932, when the veteran first experienced an unconscious attack at the age of 35. During that year he had numerous petit mal attacks in addition to epileptiform seizures which averaged about two per month in frequency. These seizures were jacksonian in type, beginning in the right foot and involving the whole body with loss of consciousness. A diagnosis of epilepsy, grand and petit mal was made. A second diagnosis of tertiary syphilis was made on the basis of a 3 plus Kahn on the blood although the blood Wassermann was negative. The spinal fluid examination showed negative Wassermann and Kahn tests, globulin negative, cells O, albumen normal, total protein 24 mgms. % and colloidal gold curve of 222211000. The neurological examination at that time was reported to be negative. The veteran was confined to a penal institution almost continuously from 1935, until 1941. While there the serological tests remained positive, although considerable antiluetic therapy was given. This therapy included 10 febrile crises with the use of malaria in 1937. Throughout this period the spinal fluid examination remained negative. No grand mal seizures were recorded during the period 1935 to 1941. However, in May 1941, the veteran had three seizures of the grand mal type. In June he was admitted to the prison hospital suffering from a right hemiplegia with motor aphasia. It was the opinion of the medical officers of that institution that the veteran had neurosyphilis in addition to an idiopathic epilepsy. This opinion was held even after a spinal fluid examination in July 1941, showed a paretic type of gold curve (4444322210), increased globulin, 62 lymphocytes per cu. mm., and negative Klein and Kahn tests.

In May 1942, the veteran was admitted to a Veterans Administration Hospital where the opinion was held that the whole neurological picture could best be explained on the basis of neurosyphilis.

A rating board of the Veterans Administration then requested that the veteran be transferred to a Diagnostic Center of the Veterans Administration in order to determine, if possible, whether or not the veteran had more than one neurological condition. In

the Diagnostic Center the veteran was examined by the Consultant in Neurology on October 6, 1942. The neurological findings at that time were those of a right hemiplegia of severe degree. The serological tests revealed a negative Wassermann with a 2 plus Kahn. The spinal fluid was clear and colorless, 2 plus Ross-Jones globulin, 10 lymphocytes, 90 mgs. protein, colloidal gold curve 0001233321, Wassermann 100% in the strongest dilution. The cerebrospinal fluid pressure was 400 mm. of fluid. AP and lateral stereograms of the skull revealed no abnormalities. The neurologist concluded that with a history of jacksonian attacks, later followed by hemiplegia, further studies should be made to rule out a brain tumor. A ventriculogram was not satisfactory and the Wassermann test on 1 cc. of this fluid was negative and the gold curve was 0001110000. An encephalogram was then recommended and performed. The neurologist agreed with the radiologist in the opinion that there was a displacement of the ventricular system to the right as from an expanding lesion on the left and further stated that it confirmed his previous impression that the veteran had a brain tumor; and he advised neurosurgery. He also concluded that the signs and symptoms were inadequate for diagnosing syphilis of the central nervous system.

The veteran was transferred to the neurosurgical service and on December 9, 1942, was examined by the neurosurgical consultant. It was his opinion at this time there was a definite blurring of the nasal margin of the optic discs with slight elevation of the right disc. He believed this represented a beginning papilledema. He concluded that the patient had a space occupying lesion in the left cerebral hemisphere (probably in the temporal lobe), with the possibility of this being a gumma but he imagined that a slow growing glioma would be found.

On December 23, 1942, a left fronto-parietal osteoplastic craniotomy was done with partial removal of a subdural granuloma with production of a large decompression by sacrifice of the osteoplastic flap. The diameter of the granuloma was 6-8 cm. and the post operative diagnosis was subdural infiltrating granuloma, involving both dura and cerebral cortex, over the pre and post central gyri of the left cerebral hemisphere. After microscopic examination the pathologist concluded that this lesion probably represented a gumma.

The principal benefit derived by the patient from the operation was the reduction in the number of seizures. He had only two seizures during the 82 days he remained in the hospital following surgery. He was transferred to a psychiatric hospital for further care and treatment and died there on September 23, 1943, exactly nine months following surgery. His death was attributed to psychosis with syphilis of the central nervous system.

Authority for autopsy was requested but not obtained.

This case is interesting because it represents a condition which is the rarest form of neurosyphilis; it is the rarest form of brain tumor; and the diagnosis could not be established with certainty prior to surgery. It also illustrates the fact that a large syphilitic lesion may be present in the central nervous system when the spinal fluid examination reveals no evidence of syphilis.

A review of the literature gives some interesting findings relating to gummas of the brain. Gummas usually begin in the meninx and penetrate the brain. This gives a point in localization, the lesion usually being cerebral and near the surface. The lesion is usually single and large. Brain tumors may be found in syphilitic individuals. Brain tumors may give a false positive serological reaction. A choked disc may occur with positive serology due to a chronic syphilitic meningitis. The usual serological combination is a positive Wassermann reaction in the blood, and a negative reaction in the spinal fluid, combined with an abnormal colloidal gold reaction and an increase of protein.

A gumma of the brain usually appears 15-20 years after the initial infection in individuals from 30 to 60. A previously resistant gumma may become vulnerable to chemotherapy after a surgical decompression. Gumma of the brain is an operative lesion.

Gummas must be differentiated from tuberculomas. The latter occur four times as frequently before the age of 20 as afterwards. They are six times more commonly

cerebellar. They are smaller and often multiple, involve brain parenchyma rather than vessels or meninges, and are not infrequently calcified. They have a better defined and denser capsule which surrounds completely caseated debris.

The diagnosis of gumma of the brain is conjectural until it is large enough to produce the signs and symptoms of a space occupying lesion. Then the diagnosis can be certain only at the time of operation. With the more effective antibiotic therapy of syphilis in recent years, gumma of the brain will become more of a rarity than it is at present and may no longer require surgery for its eradication.

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PUBLIC HEALTH SERVICE GRANTS

Announcement is made of new research grants recently awarded by the Surgeon General of the Public Health Service upon the recommendation of the National Advisory Mental Health Council at its June meeting. The names of the principal investigators for each project, their location, the subject of the study, the period for which the project is approved for grant support, and the amount awarded for the first year are enumerated. Twenty-five new grants were awarded totaling $373,178.

Anderson, John E. and Harris, Dale B. University of Minnesota. The relation of childhood behavior to adjustment in adulthood. Three years. First year $14,796.

Bandura, Albert. Stanford University. Relation of therapeutic competence to personality of psychotherapist. One year $1,851.

Blocksma, Douglas, Fox, Robert, and Lippitt, Ronald. University of Michigan. A study of methods for improving social acceptance and participation of rejected children. Three years. First year $27,972.

Blom, Gaston E. and Long, Robert. Massachusetts General Hospital. Study of emotional factors in children with rheumatoid arthritis. Four years. First year $17,981.

Busse, Ewald W. Duke University. Effect of aging upon the central nervous system. Two years. First year $14,534.

Cohen, Louis D. Duke University. The assessment of change in anxiety level. One year $5,400.

Dean, John F. and Williams, Jr., Robin M. Cornell University. Social and cultural factors in adjustment of American women. One year and a half. First year $10,770.

Drake, Miles E. The Vineland State School. Metabolism of phenylpyruvic acid in mental deficiency. Three years. First year $15,869.

Fink, Maximilian. Hillside Hospital. Perceptual changes induced by drugs and electroshock. Two years. First year $9,612.

Gerty, Francis J., Garner, Ann Margaret, and Wenar, Charles. University of Illinois College of Medicine. Studies of psychosomatic disorders in children. Two years. First year $22,396.

Haggard, Ernest A. The University of Chicago. Family adjustment, personality, and mental processes. Two years. First year $14,018.

Harlow, H. F. University of Wisconsin. Symposium on interdisciplinary research in the behavioral, biological, and biochemical sciences. One year $8,910.

Indexing Psychiatric Interviews

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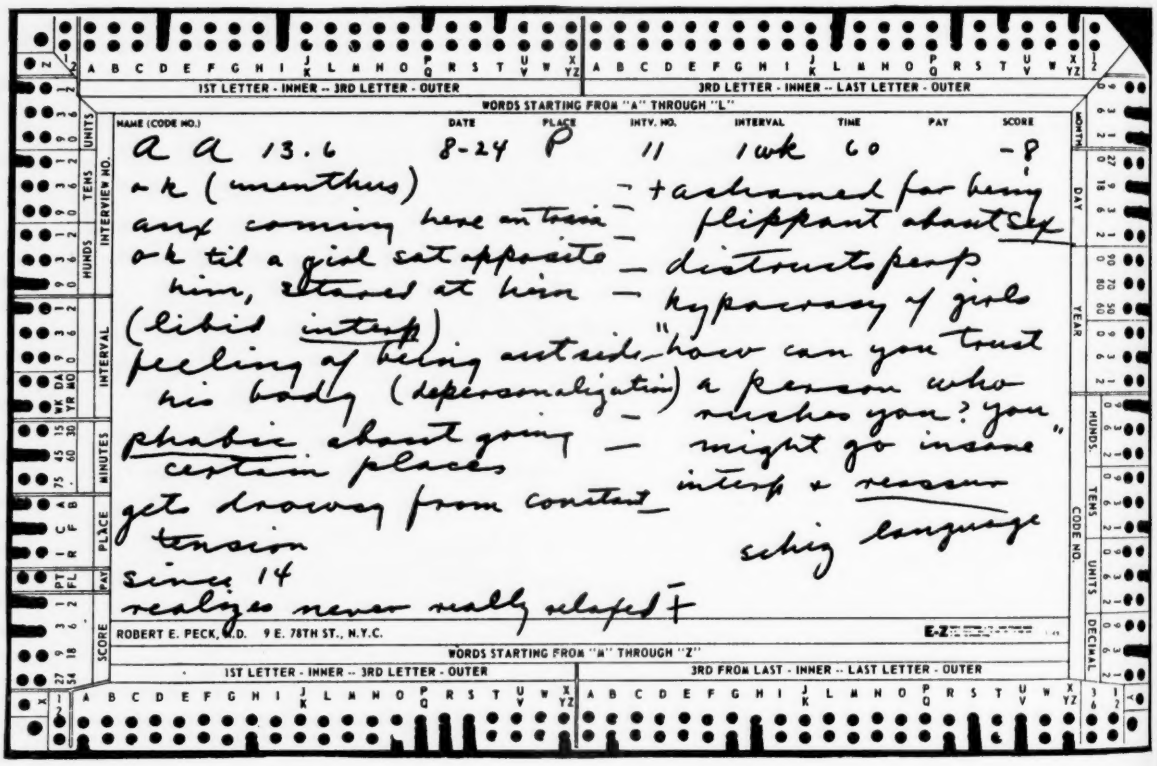
From time to time in the psychiatric literature there appear wistful expressions of a need for a more systematic and statistical analysis of the psychotherapeutic process.[1,2] Lately a number of theoretical papers dealing with the problem have been published and some practical applications demonstrated.[3-11] This is a trend we may confidently expect to grow. Technical difficulties are the greatest deterrent. I propose in this paper to describe a tool that may help to stimulate further work along these lines.

Psychiatric interviews are the vehicle through which most clinical material is gained and is the most widespread approach to treatment. Scientific correlating of the material calls for accurate recording of these interviews.[12] However, even where the technical facilities for this are available, the resulting accumulation of data becomes formidable to read and difficult to assimilate. Obviously, some type of indexing is indicated. My own attempts at recording interviews early made me aware of its importance. A simultaneous interest in punched cards made me explore their use for this purpose. The card described here represents the culmination of many months' experimentation and I have used it long enough to demonstrate its practicability and reliability. There is nothing original about this technique for the principle of edge-punched cards has been in use for a number of years,[13] although this is the only adaptation to the raw material of the psychiatric interview that I know of. The card uses a combination of direct, geometric, and word coding. The origin of direct coding is attributed to an Englishman named Perkins.[14] Geometric coding of the type used here is a variation on the usual 1, 2, 4, 7 coding and was introduced by R. L. Hollingsworth.[15] Word coding was invented and developed by Carl S. Wise.[16] The technical details of punching and sort-



ing edge-punched cards can be obtained from Casey and Perry's source book on the subject, from the manufacturer of the cards, or from a previous paper of my own.[17]

Fig. 1 shows the index card coded and punched after an interview. It is a 5 x 8 inch edge-punched card of moderate cost. Across the top and bottom of the card are duplicate alphabets which are for the word coding. Notes are taken on the front and back of the card during the interview and after it is over, the essential aspects of the interview to be recorded are either underlined in red pencil or written out in the margins. These words or phrases are then coded and punched in the top or bottom of the card by using the following conventions: words having the first initial in the first half of the alphabet are punched in the top two alphabets, those with the first initial in the last half are recorded at the bottom. The first letter of the word is punched in the inner hole, the third letter in the outer hole of the left hand alphabet; the third from the last letter is punched in the inner hole of the right hand alphabet, while the last letter is punched in the outer hole. The same procedure is followed for short phrases. Dropping the suffixes serves to increase the selectivity of the process. For example, the word *reactive* is coded *react* as it is the common part of the different words *reactive, reaction, react,* etc. and would be punched *r* deep and *a* shallow on the left of the bottom of the card, *a* deep and *t* shallow on the right. Final *e* and *es* are dropped routinely. Thus any desired aspect of the interview can be indexed and selected from a group of similar cards by the simple device of passing four needles through the appropriate holes and lifting the deck. This makes the card a versatile means of recording any type of record such as abstracts, notes, or experiments, as well as interviews, as it eliminates the need for a fixed direct code and allows for great variation in the choice of items that one may need to record. It loses somewhat in efficiency because of the false drops, a phenomenon which is not present in direct coding, but this is a minor matter when compared to the almost unlimited scope it allows. In any case, false drops are easily eliminated by inspection of the dropped cards.

The sides of the card are given over to the coding of specific and standardized information about the interview. Each patient is assigned a code number which is punched in the lower right hand corner. Up to ten thousand patients can be accommodated by the space allowed. Geometric coding is used. Thus a patient whose number is 985.3 would be punched 9 in the hundreds column, 6 and 2 in the tens column, 3 and 2 in the units column, and 3 in the decimal column. This method of coding patients requires a master file of all patients with their respective numbers. I have found a circular file handy for this. An alternative method that would not require this is to assign a number to each letter of the alphabet as is done on the left side of the card in the category marked *place* (alphabetic coding). Then the name can be punched directly on the card by using the initial of the first name, the first, third, and last letters of the last name. The first method is preferable as it eliminates any possibility of overlap between patients, and, by sorting the cards numerically, they automatically are sorted alphabetically.

The date of the interview is punched in the upper right hand corner. If the date were June 9, 1953, it would be punched *6* in the months column, *9* in the day column, *50* in the decades column, and *3* in the units column.

The number of the interview is punched in the upper left hand corner. The fifth interview would be punched *3* and *2* in the units column, *0* in the tens, and *0* in the hundreds column. This allows for up to a thousand interviews on any patient.

The interval between the present and the preceding interview is punched in the next lower category. Here direct coding is used to indicate the unit of measurement. An interval of a week would be punched *1* and *wk*.

Direct coding is used also for the length of the interview. A 40-minute interview would fall between 30 and 45 and would be punched under 45.

Alphabetic coding is used for recording the place of the interview. I use the first initial of the clinic. If this were *P*, it would be punched *I, F,* and *A*. If two places have the same first initial, a synonym for one of them is used.

Payment for private interviews is recorded in the next category. A full payment is punched deep, part payment shallow, and, of course, no payment, unpunched. This facilitates bookkeeping.

The category marked *score* is for the experiment of assigning a numerical estimate to each interview, and might conceivably be useful for purposes of comparison or correlation. The method I have experimented with, although crude, is based on what I conceive to be the way value judgments are arrived at—the addition of good or bad signs. The numerical figure obtained is punched in the numerical columns and its sign utilizes the corner hole marked *X*. If the number is positive (the minority), the *X* is punched, if negative, unpunched. Considerable spread is found in scores and, as might be expected, they show a normal distribution, somewhat skewed towards the negative side. An attempt to compare this score with estimates of the interview as a whole (N 406) graded as follows: 3, best; 2, better; 1, good; 0, neutral; —1, bad; —2, worse; —3, worst, gives a correlation (contingency) of .441.[18] By having enough of these cards (and they accumulate at an alarming rate), it is possible to compare interviews which have been selected as alike in every way except content, and these can then be studied for any similarities they may show. The card seems to be applicable to many simple statistical procedures as well as for indexing.

At first glance this method of indexing may appear to be a formidable procedure. It is, however, extremely simple and with a little practice it becomes almost automatic. I usually code the card immediately after the interview. This takes at most only a minute or two. The punching time averages 1½ minutes per card. This detail can be done by a secretary or sent to an agency as the card is not identifiable without the master patient file.

I believe that by using this indexing tool interviews can be made more scientific and that by lessening the tendency to generalize from one or two observations, our thinking may become more accurate. Psychiatrists have pointed out the fallibility of the human memory, but they are often the worst offenders in this, when analyzing their clinical experience. Memory tends to bring back only one or two instances of a coincidence of facts and to ignore the other instances where they did not occur together. Punched cards will bring them all back.

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Electroconvulsive Therapy and Depressions

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Introduction

Since the introduction of electric shock to produce convulsions in mental patients in 1938, by Cerletti[1] and Bini of Rome, this method has grown rapidly in spite of its formidable title and prejudice against it by those within and without the medical profession. While one-third to one-half of psychiatric literature today has to do with ECT (electroconvulsive therapy), there have been few articles published for the non-psychiatric physician. It would appear that those working with ECT have been content to let the method stand or fall on its own merits. Now that ECT has been in use more than ten years, and is used by ninety per cent of the psychiatric hospitals over the country, it is timely to discuss more frankly not only the prejudice but also the indications, contraindications, complications, and results of this relatively new therapy.

Much of the prejudice and suspicion against ECT is no more than the prejudice and suspicion which exist in people's minds concerning any new method. As a boy, and even as a general physician in a rural community twenty years ago, I remember lay people expressing their ignorance and suspicion of surgery. "If the Lord had not intended us to keep our organs (referring to a septic appendix) he would not have given 'em to us," was an example of this thinking. Recently, when a patient advised me that her former doctor stated that he "did not *believe* in electric shock treatment," and I learned that he had never even witnessed one such treatment, I was reminded of those early views which some lay people held concerning surgery. In spite of the occasional prejudiced physician concerning ECT, non-psychiatric physicians as a whole have welcomed this new therapy, often more enthusiastically than have some psychiatrists. One doctor, for instance, whose patient had spent 18 months and several thousand dollars taking psychoanalytic treatment, was astounded and gratified at the improvement in his patient obtained by only five ECT's.

ECT should not be confused in any respect with ancient methods of shock or pain, such as throwing patients in cold water, or frightening them with fire or torture, because the patient receiving ECT loses consciousness instantaneously, so that he feels no pain whatsoever. Since there is no shock in either a physical or psychic sense, the term electroshock is misleading and should be abandoned for "electrosleep" or "electroconvulsive therapy." Indeed, it is not unusual for a patient who is recovering from an ECT to contend that he has not had a treatment, because he has felt nothing and he has a complete amnesia for the treatment itself. Just how ECT does produce improvement in mental patients will be discussed subsequently.

Indications for ECT

The outstanding indication for ECT is melancholia or pathologic depression. A depression is not always easy to recognize; in the first place, the physician is usually in search of an organic entity and often fails to consider depression in the differential diagnosis; in the second place the patient does not like to admit that he is depressed. In fact, a patient may look despondent, cry all day, become obsessed with morbid subjects, even attempt suicide, but deny being depressed. Sometimes a patient may admit melancholia in some other terms but avoid the word "depression." He may admit being "low in energy," having a "lost or hopeless feeling," being "disgusted with myself," or having an "unworthy feeling," or feeling "like I've lost out with the Lord," but still not admit feeling depressed. If the physician has this condition in mind in differential diagnosis, however, he will be impressed by one of these expressions. The patient may admit being worried or depressed over some physical ailment, thus diverting the physician's attention away from the true illness, but vehemently deny being depressed without any apparent cause. If the patient feels

it necessary to give a long explanation for his depression, the physician should suspect a pathologic mood. It seems to be the spirit and desire of man to keep his emotional flag flying high and if he does slip in this respect he likes to have an acceptable cause; but the physician must keep in mind that for pathologic depressions no cause in the environment is necessary. In most instances there will be found a history of previous depressions, either in the present patient or in other members of his family, or both.

There are certain physical symptoms, such as fatigability, stubborn insomnia, menstrual changes, anorexia, loss of weight, tachycardia, inability to get a deep breath, headaches, gastric distress and vasomotor disturbances, which accompany almost every depression and should be considered a part of the illness.

It should not be assumed that all patients who are depressed are in need of ECT. A great many of the milder cases can be treated by the general physician, internist, or psychiatrist, in his office by conservative measures. However, when the depressed patient's symptoms preclude rest, sleep and nourishment, ECT should be considered. As long as a patient is able to eat, sleep and work, certainly the physician is justified in continuing office treatment. When a depressed patient is exhausting himself because of agitation, cries excessively, is subject to fits of temper, is threatening homicide or suicide, is drinking to excess, using drugs, or is reading and preaching the Bible in a fanatical manner, ECT should not be withheld. While ECT has its dangers, which will be discussed, it should not be forgotten that psychosis also has its dangers, to say nothing of the time lost and money spent, not only by the patient but also by other members of the family. The psychiatrist, like other physicians, has a natural tendency to use conservative measures whenever possible, resorting to ECT only in those cases in which the psychosis threatens to bring havoc upon the patient's life.

Patients with morbid delusions, especially regarding death, delusions of guilt, remorse, and patients with "Holy Ghost" sort of religious obsessions, usually obtain good results with ECT. Patients with a great deal of autonomic distress, such as severe headache, "migraine," vomiting due to pylorospasm, "colitis," tachycardia, intractable urticaria, marked insomnia, associated with a melancholic mood, usually obtain good results, and sometimes a miraculous remission, with ECT. Speaking of somatic symptoms relieved by ECT, I have seen the pains of a phantom limb, severe itching and burning of an intractable neurodermatitis, urinary retention due to nervous tension, gaseous distention of neural origin which had been diagnosed intestinal obstruction, relieved by means of ECT. It should be emphasized that neither neurodermatitis nor other somatic complaints, in the absence of a depression, is an indication for ECT.

The best results from ECT can be expected in acute illnesses, patients with marked depressions and acute autonomic dysfunction. Although patients who have been ill a long time and who fall into the hypochondriacal category, may not obtain as good results, even these are often helped by means of ECT and should have a trial with this treatment. ECT is not indicated in psychoneurosis but it should be remembered that many patients given a diagnosis of psychoneurosis are really suffering with mild manic-depressive psychosis.[3] Indeed, some of these patients are often treated for months by psychotherapy, a method which is intended to help psychoneurotic patients, but which has no effect upon an endogenous depression.

Everyone agrees that ECT is not indicated in the treatment of chronic alcoholism per se, but there are a good many alcoholics, especially dipsomaniacs, who drink as a result of episodic endogenous depressions. When studied closely as to his (or her) mood swings, attitudes, and reaction to alcohol, it can usually be established that this type of alcoholic is basically a manic-depressive personality. We have obtained some excellent results by means of ECT in this type of chronic alcoholic, the electroconvulsive therapy removing the melancholia or levelling off the severe mood swings which predisposed the individual to alcoholism. A recent article by Cleckley[2] et al. points out the advantageous use of ECT in relieving the withdrawal effects in narcotic addicts.

The psychiatrist who uses ECT looks upon

it as an auxiliary agent and does not fail to institute other measures necessary for the rehabilitation of the nervous or mentally ill patient.[3] In fact, as previously indicated, in some instances it is necessary to give ECT first to the disturbed individual in order to get him relaxed so that more conservative measures may be employed. Briefly, these measures include the following: explanation of the patient's somatic symptoms so that he won't wear himself out looking for a physical diagnosis; removal of stress and strain in the environment, factors which act as precipitating or aggravating agents; teaching the patient how to rest and relax and to live within the margin of his own peculiar nervous reserve; providing suggestions, reassurance and friendly counsel to the susceptible individual; advice to family and friends concerning the nature of the patient's illness and what they can do to help or hinder his recovery; the proper diet and the proper and supervised use of drugs and vitamins; occupational therapy. Just to give ECT and not to carry out these other important procedures of mental hygiene would be negligence indeed.

Contraindications to ECT

Coronary disease, aortic aneurysm, angina pectoris, brain tumor or abscess, and glaucoma are considered definite contraindications to the use of convulsive therapy. Back in 1940, hypertension, cardiovascular disease, advanced age, arteriosclerosis, pregnancy, tuberculosis and any deformity of the spine were considered to be contraindications to the use of ECT. We now know, however, that none of these conditions constitute an absolute contraindication to this treatment. In fact, experience has shown that psychiatrists have been unduly conservative in the use of ECT.[4] Hypertension, once considered a contraindication, is now often found to be only a complication of the nervous condition, and frequently improves as a result of ECT. Cardiovascular disease is still a relative contraindication but even here the dangers of the treatment must be weighed against the danger of the stress, strain and exhaustion resulting from the patient's psychosis. An agitated patient with cardiovascular disease may be doing himself more harm untreated than if he were given ECT. Only experience and careful study of each individual patient can decide this issue. Kalinowsky[5] stated as follows concerning the contraindications to ECT: "It bears repetition that the seriousness of the physical illness and of the mental condition must always be carefully weighed against each other. However, pre-existing physical diseases should not be overstressed as contraindicative of a treatment which, although admittedly drastic, results in surprisingly few ill-effects on the organism as a whole."

Advanced age has long ago been discounted as a contraindication to the use of shock therapy.[6,7,8] Again, the dangers of the patient's agitation, malnutrition, combativeness or threats of suicide, must be weighed against the dangers of ECT. Fortunately, elderly patients take shock therapy well and, because of the weaker and flabbier musculature, they have milder convulsions and fewer complications than do younger, stronger patients. Mournfully depressed, elderly patients, agitated, wearing themselves out as well as their families, often calm down nicely with only four or five ECT's. Indeed, some of the most remarkable remissions from mental illness in our experience has occurred with elderly individuals. Incidentally, ECT has proved the reversibility of mental illness in the aged, conditions once thought to be entirely organic and irreversible. In addition to cerebral arteriosclerosis and senile changes, therefore, we now know as a result of ECT, that other physiologic disturbances are occurring in these elderly patients. In this respect, as well as in others, ECT has not only proved itself to be a valuable therapeutic agent, but it is also opening up new fields for research in psychiatry.

Depressed patients who happen to have tuberculosis have been treated satisfactorily with ECT without aggravating the pulmonary disease. In fact, patients in an agitated depression can cooperate more efficiently to the rest, sleep, and improved nutrition necessary for the treatment of the tuberculosis, after ECT has soothed the disturbed mental illness.[9]

Ten years ago it was thought that pregnancy would certainly be a contraindication

to the use of electroconvulsive therapy. In the meantime a number of workers have reported on the use of this treatment during pregnancy. The writer has given ECT to patients at every month of pregnancy without producing any uterine contractions, bloody show, or other obstetric complications. This has been an encouraging advance in the practice of obstetrics as well as psychiatry, because patients with mental illnesses during or following pregnancy are often the most suicidal patients with whom we have to deal. In fact, the successful use of ECT in psychotic patients who are pregnant has required a new consideration of the indications for terminating pregnancy in such patients. Prior to the use of ECT in these patients therapeutic abortion, which really accomplishes nothing insofar as the mental illness is concerned, was not infrequently resorted to. Today, most psychiatrists take the view that the pregnant psychotic patient should first be given ECT.[10] Even if this treatment has to be repeated at term, in case of a recurrence in the psychosis, it is safer and much to be preferred to the abortion. The latter often only increases the patient's feelings of guilt and, as stated, it does nothing to relieve the mental illness. Patients who are known to be manic-depressive, or who have suffered with a post-partum psychosis following a previous pregnancy, and who already have two or three children, may be considered for sterilization at the time of delivery but, since we now have ECT, abortion should be deferred for the most extreme or unusual cases.

Weak and debilitated patients, unable to eat and sleep, are not contraindications for ECT. In fact, the best way to build up a debilitated mental patient, provided convulsive therapy is indicated, is to proceed at once with this therapy.[5] Since the debilitation in these patients is secondary to the autonomic dysfunction which interferes with proper nutrition and assimilation of calories, reorganization of the physiology by means of ECT is an essential part of the physical rehabilitation.

Mental illness caused by or complicated by bromide intoxication is made worse by ECT.[11] Before giving this treatment, therefore, the psychiatrist must be sure that all bromides have been eliminated from the system.

Intracranial lesions, such as tumor or abscess, or Pick's disease, which is a frontal lobe sclerosis, are definite contraindications to the use of ECT. In fact, the only adverse results observed from ECT occurred in patients either with bromide intoxication or a cerebral lesion.

While there are only a few absolute contraindications to the use of shock therapy, the writer is particularly cautious in giving the treatment to patients with a *combination* of organic illnesses, such as cardiovascular disease in the presence of diabetes, or cardiac hypertrophy and atelectasis. Regardless of the physical condition, the psychiatrist must weigh the dangers of the psychosis, such as suicide, starvation, or exhaustion from agitation, against the dangers of the therapy. While the psychiatrist will often have to consult with an internist or surgeon in determining whether or not a patient should be given ECT, the ultimate decision is up to the psychiatrist because only he is familiar with the actions and reactions of the treatment. Generally speaking, a review of the literature and present practice will show that psychiatrists have been unduly conservative in the use of this treatment, formerly withholding it in many cases because of only relative danger when the chance of benefit far out-weighed the risk.

Bone conditions as contraindications and bone fractures as complications will be discussed in the next section.

Complications of ECT

There is a great deal more to the administration of ECT than simply giving the treatment. The family must be educated, not only to the nature of the illness being treated but also to the results and side effects of the treatment. The family of each patient is advised of the possible complications of the treatment but almost without exception, having dealt with the problem of the psychosis, they are anxious for the patient to obtain a treatment which promises specific relief. As previously stated, most nonpsychiatric physicians have appreciated the results of ECT and render a most helpful service in educating and obtaining the

cooperation of the family. Most of the complications of ECT are on the order of nuisances but require considerable patience on the part of all concerned in the welfare of the patient.

During and following a series of ECT's there is always a relative and transient impairment in memory. Usually of minor importance, this complication is the chief reason for keeping the patient quiet, free from visitors and responsibility for two weeks following the shock therapy. In fact, each family is given a set of directions, concerning what the patient should and should not do during the period of convalescence. Since the patient is feeling so much better he is anxious to see people, talk, go to church or to town, or do other things which, in his convalescent state, might embarrass him later or cause a recurrence of his illness. The patient and his family can be assured that no serious or permanent impairment of memory, or brain damage, occurs as a result of this treatment. While it is desirable to give as few ECT's as necessary in order to obtain results, cases[12] have been reported of patients who received as many as a hundred or more ECT's without suffering any intellectual deficit. Incidentally, impairment of memory is one of the most frequent bugaboos held by the layman, and even by some physicians, concerning ECT. Most patients find this fleeting complication to be of no more than humorous interest. It is true that patients are often unable to remember details of their illness, or events occurring during the time they were under treatment, but this is as often as much an advantage as it is otherwise.

Among the physical complications of ECT, least important are the muscular strains, more rarely torn muscles, which result from the tonic phase of the grand mal seizure. Such complications as circulatory shock, prolonged apnea or coma, following a convulsion, occur rarely and may require artificial respiration, oxygen and stimulants.

While dislocations of the jaw or shoulder (easily reduced while the patient is still unconscious) may occur occasionally, the most important complication of ECT are bone fractures, particularly compressed fractures of the bodies of the dorsal vertebrae. By hyperextension of the spine during the treatment and proper restraint, most of these fractures can be prevented. However, they do occur and must be accepted as one of the risks of this therapy. It should be noted, however, that compressed fractures of the bodies of the dorsal vertebrae cause very little disability, only a transient pain, and no deformity or neurologic sequelae. Polation and Linn[13] followed for ten years twenty-four patients who had sustained compressed vertebral fractures, and who had not received any special orthopoedic treatment. Only four of the twenty-four patients complained of back pain ten years later, the remaining twenty patients having no pain referable to the back. There had been no neurologic sequelae and no progression of the orthopoedic defect, as indicated by x-rays and other examinations. Incidentally, some of these twenty-four patients had received a course of ECT's subsequent to the one in which the fractures occurred. One of the writer's patients returned to his work riding a bicycle within a few weeks after suffering compressive fractures of the bodies of the 4th and 5th dorsal vertebrae. If the physicians and nurses who see these patients will refrain from using the term "broken back" considerable disability can be prevented. If the spines of a series of epileptic patients were x-rayed a number of asymptomatic vertebral fractures would be found. A recent article by J. Gershon-Cohen, et al., in the Journal of American Medical Association,[14] describes thirty-five patients who had suffered vertebral fractures which were untreated and which produced no symptoms. While every precaution should be taken to prevent such fractures it is a noteworthy fact that they have caused little subsequent difficulty.

Fractures of the hip, much rarer than the vertebral fractures, may occur with ECT. Incidentally, in patients who have suffered a fracture, or in individuals known to have susceptible bones, Anectine (Succinylcholine Chloride) may be given intravenously with pentothal sodium immediately preceding the ECT and thus prevent the muscular contractions and without interfering with the beneficial effect of the convulsion. The writer

does not consider curare a safe drug to use routinely in ECT.

To those who seem to see only the complications of ECT, it should be reiterated that fractures are unusual when proper precautions are taken and when they do occur they are usually of minor importance. ECT is not a treatment which should be administered without serious consideration but, on the other hand, only the timid and inexperienced would withhold a treatment which offers so much relief to such unhappy patients. It should be remembered that complications of a psychosis, such as family quarrels, divorce, prolonged illness, homicide, suicide, or commitment to a state hospital, may be much more detrimental to a patient or his family than the possible complications of ECT.

There is really no quarrel between the psychiatrist using psychotherapy and the one who uses ECT, because the psychotherapist deals with, or should deal with, patients who are only mildly nervous or depressed, patients usually labelled psychoneurosis, while the psychiatrist using ECT has to treat patients who are disturbed, psychotic, incapable of benefiting from a purely psychologic method. It is an obvious fact that if such patients could be relieved by psychologic methods, electroshock therapy would not have become so widely used as it is today. As previously indicated, after a remission is obtained in the psychosis by means of ECT, a number of conservative measures, described in detail elsewhere,[3] must be instituted in order to complete the rehabilitation of the patient.

Technique and Results of ECT

Since electroshock therapy has become a procedure of such widespread use, the Committee on Therapy of the American Psychiatric Association has set up standards to follow in its administration. In order to prevent fractures and dislocations it is recommended that the patient's spine be placed in hyperextension and that the patient be loosely restrained. ECT produces a generalized convulsion, lasting 40 to 50 seconds, and having the tonic and clonic phases characteristic of a grand mal seizure. As previously stated, most patients do not mind the treatment because they feel no pain or electricity and also because after the first treatment, they are less tense, nervous, and apprehensive. Patients in a moderate depression are usually given five or six treatments on alternate days, although the series must sometimes be repeated because of a recurrence in the illness. While it is desirable to have the patient in the hospital for this treatment, ECT may be given on an out-patient basis, provided the patient and his family will follow directions. We have patients travelling 60 to 75 miles for the treatment on an out-patient basis but insist that they remain quietly at home at other times, strictly avoiding crowds, company, or church. Patients whose psychosis is complicated by alcoholism, family disputes, or excessive preoccupation with religion, are urged to remain in the hospital for ECT.

Observation of a patient during a course of ECT's is indeed an interesting thing to behold. The changes are as conspicuous in the physiology of the body as in the emotional or psychic sphere. The thick, splotchy skin with obstructed glands, so often present in depressed patients, smooths out into a clear and more normal texture; the patient smiles, sleeps better, and he begins to relish food, often for the first time in months. On the mental side it is noted that the patient has ceased to be depressed, that he takes more interest in his environment, and that he has stopped worrying and agitating about the obsession, whatever it was, that brought him to the hospital. It is often stated that suicide is almost unknown in a patient undergoing ECT. So dramatic and objective are these results with ECT that the therapist is reminded of the other great specifics in medicine, such as neoarsphenamine in syphilis or penicillin in pneumonia. Various workers have called attention to the fact that the improvement in autonomic functions of the body parallel the improvement in mood, indicating that whatever physiologic disturbance causes the somatic symptoms also causes the depression. This is an important lead in pychiatric research which has been uncovered by ECT.

Spontaneous remarks made by various patients, following ECT, graphically illustrate the improvement in both psychic and physi-

cal functions: "I feel like a different person"; "It seems as if I've been dead and brought back to life again"; "Life looks real again"; "I feel more like my normal self than I have in years"; "I don't know what you did to me but whatever it was my body and mind work better now". These and many other similar statements by patients themselves indicate that truly a specific and substantive change has occurred in the organism.

Incidentally, because of the confusion incident to the treatment, psychotherapy, except for guidance and reassurance, is deferred until two or three weeks following the series of treatments.

While some writers state that 90% of the patients in a depression are cured by means of ECT,[15,16,17] this figure is probably exaggerated. The writer has estimated that 75% of the patients in a depression treated by ECT show 75% improvement; there are some patients who manifest a miraculous remission of all symptoms while there are others who are improved between 50% and 75%; there are a very few, probably less than 5% who are not improved at all, and less than 1% who are made worse by the treatment. The latter can often be shown to have bromide intoxication or some organic disease of the brain and were therefore not proper candidates for shock therapy. In view of the natural remissions in depressions, as well as the frequent spontaneous recurrences, an accurate estimate of the results of any treatment is difficult to establish. However, ECT is now recognized in all psychiatric hospitals as the best treatment ever known for endogenous depressions.

ECT has not only made a record in its phenomenal results in individual patients but, to a great extent, it has, more than any other one thing, changed psychiatry from a purely institutional procedure to an active, dynamic branch of medicine. Numerous patients who formerly would have had to go to a state hospital can now be treated under private auspices and often at a very moderate expense.[18] This is a fact of such great importance socially, economically, vocationally and industrially, that the value of ECT would be impossible to estimate. When an illness, which formerly often required two or three years hospitalization, can now be relieved in that many weeks, one may obtain some idea of the over-all economic value of this treatment. Patients are being discharged earlier from the state hospitals and the latter, as a result of ECT, are able to care for more patients with less personnel and equipment. Physical restraints are almost unknown in mental institutions today and the destructive, homicidal patients can at least be sedated with convulsive therapy so that they can be cared for with much less danger and expense.

How ECT Works

Even more important than these immediate and individual results from convulsive therapy, ECT has opened up an entirely new field for psychiatric research. Those who would attribute the results of ECT to suggestion, shock or fright, or even to the transient impairment in memory, take a very superficial view of convulsive therapy. In 1942 Temple Fay[19] wrote an interesting article, called "The Other Side of a Fit," which reminds us that the visible signs of a convulsion tell us nothing concerning the chemical changes taking place in the brain. Once we learn how ECT works, or what occurs in a fit or convulsion to alleviate mental disturbances, we may discover just what insanity is. If we can find out just what occurs in shock therapy, the next step, of course, will be to perfect a less drastic agent which will produce a similar change. In 1943 Foster Kennedy[20] stated that the artificially produced convulsion tends to correct the sympathetic-parasympathetic imbalance occurring in depressed patients, a statement which recalls a previous one made by Douglas Singer,[21] that manic-depressive psychosis should be referred to as a "periodic autonomic imbalance." Other writers, such as Tanner,[22] Gellhorn,[23] Kalinowsky,[5] Funkenstein,[24] et al., and Royce,[25] et al., have observed and emphasized the autonomic dysfunction in these patients and its reorganization by means of ECT. Hench,[26] Altschule et al.,[27] Pincus and Hoagland,[28] have stated that the results of ECT are produced through the hypothalamus; there is stimulation of the adrenal cortex with an increased production of adrenocorticotropic hormone. Royce et al.[25]

reported a significant increase in adrenal weight in rats following ECT. Selye and Fortier,[29] who consider that psychoses may result from adrenal insufficiency, also feel that shock therapy acts by stimulation of the pituitary-adrenal mechanism. Somewhere, locked up in the secrets of the hypothalamus and its reciprocal relationship with the cerebral cortex, as well as with the pituitary and adrenal glands, are to be found answers which may revolutionize psychiatry.

Clinically, it is understandable that the results of ECT are produced through its action on the hypothalamus because the many autonomic disturbances, such as headache, pylorospasm, sleep disorder, abnormal metabolism, menstrual irregularities, and faulty skin physiology, occurring in depressions, are often cleared up completely by this therapy. It is well known that most of these functions are centered in the hypothalamus where is also located the emotional center.[30] It is conspicuous also that these physiologic manifestations of a depression are improved in the same proportion as is the mood disorder, emphasizing over and over that the autonomic disturbances and the mood disorder have a common origin, most likely in the hypothalamus.

Anyone can take a look into the history of psychiatry and see that every psychiatric disease for which we know the cause, there will be found a toxin, an organism, or some physiologic or organic disturbance. In other words, mental illness, as far as we have been able to determine, is caused by the same agents which cause other forms of disease. Paresis is a complication of syphilis; dementia of pellagra is secondary to a vitamin B deficiency; arteriosclerosis and senile plaques in the brain account for mental deterioration, and schizophrenia has been shown to be accompanied by a low anterior corticotrophic hormone response to stress.[28,29] Now, manic-depressive psychosis, which is one of the most prevalent of all psychiatric diseases, is found to be specifically effected by convulsive therapy. All of this is not to say that psychologic methods will not always be useful and necessary in the practice of psychiatry, but it does indicate that there has been considerable neglect of the organic and physiologic factors involved in mental illness.

In conclusion the writer wishes to quote a paragraph from Gordon, one of the many who has recognized the importance of this relatively new therapy. He states: "Electroshock therapists were heckled by the timid, the stubborn, the skeptics and the conservatives who foretold irreversible injuries of the brain, fractures, relapses and even fatalities—but they were not discouraged. Particularly receptive to this new method were those on duty within crowded mental institutions, moving through wards of humans seemingly doomed to incessant inner turmoil, to regression down to the level of a dumb animal or even to the vegetable stratum; mute, motionless, filthy and slowly deteriorating or belligerent and self-destructive. These workers who don't deal with a few selected psychoneurotics in their offices or with chosen 'demonstration' samples presented in a university lecture hall, but with thousands upon thousands of wretched lives, for whom prolonged individual attention is impossible, could not resist grasping any straw of salvation given to them. And the phenomenal results obtained by them in the treatment of a grave human affliction, against which physicians, up to 1938, were as helpless as Hippocrates was 2300 years before them—could not deter the pioneering spirit from following a new path."

Thus psychiatry, which before ECT was bogged down in questionable and often fantastic psychoanalytic interpretations, today is seeking through physiology, chemistry and the endocrines the answers which failed to develop out of purely psychologic studies. Electroconvulsive therapy, which is a valuable treatment even in individual cases, may lead to the secrets which have baffled mankind for centuries.

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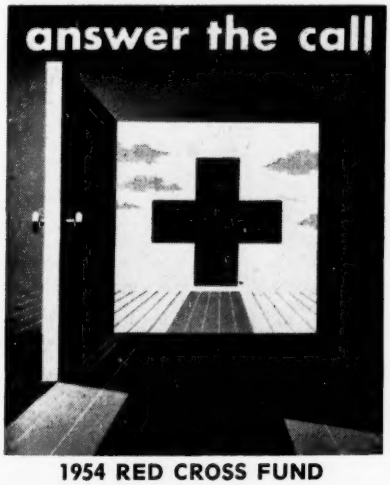

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A Study of the Relationship Between Music and Post-Electroshock Awakening

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Problem

This reports the results of an investigation into the effect of music upon the speed of awakening after EST. It has been observed by musical therapists that the length of time for awakening after the administration of EST varies with the kind of music the patient hears. Leedy and Leedy[2] noted that the use of music with EST resulted in more rapid return of consciousness and decreased restlessness. Price et al.[3] employed music that was melodic and sentimental, without strong rhythm, in the immediate post shock awakening phase "to elicit pleasure and gradually arouse a desire for voluntary activity." Altshuler[1] found that the most regressed psychiatric patients could sometimes be reached by simple rhythmic patterns. Viewing the immediate post-convulsive state as a physiologically induced regression, we became interested in the effect of musical rhythm on patients recovering from EST. A research design was therefore organized to test the following hypothesis: Speed of awakening after EST is related to the presence and type of music played during the administration of the treatment and during recovery.

Procedure

The experimental design was as follows:

	Monday	*Wednesday*	*Friday*
First week	Silence	Music A	Music B
Second week	Music A	Music B	Silence
Third week	Music B	Silence	Music A

Music A consisted of smooth orchestral arrangements of classical and lighter quiet selections, such as Tchaikowsky's "Andante Cantabile," Kreisler's "Liebeslied," St. Saens' "The Swan," and "Beyond the Sea." Music B consisted of bright, loud, jazz arrangements with selections such as Wildroot, Limehouse Blues, Hobnail Boogie, Jungle Drums, by popular bands—Woody Herman, Count Basie, Gerry Mulligan, and small Dixieland groups. Shock treatments were administered with a standard apparatus using both instantaneous and glissando technique, unmodified or modified by muscle relaxants, but constant with each patient. Pre-medication consisted of atropine and, in the cases in which muscle relaxants were used, intravenous sodium pentothal. No other sedation was administered and above sedation was also constant for the individual patient. Patients received an injection of atropine at 8:00 a.m. and then proceeded to the EST waiting room whence they were summoned individually for treatment. There was no pre-shock music. EST was administered in an area screened from the remainder of a large dormitory. The latter constituted the post-shock recovery area. Music was played from tapes on an Ekotape recorder. Administration of EST was usually completed by 9:30 to 9:40 a.m.

Prior to this investigation a meeting was held with the nurses and attendants of the EST team. The method and purpose of the study was explained and possible sources of error were discussed. An individual meeting was held with the attendant assigned to the EST recovery area and "awakening" of the patient was objectively defined. It consisted of two parts: (1) the point at which the patient showed signs of awakening—opening his eyes, glancing about, sitting up; (2) the point at which the patient actually did leave the recovery room.

A card was prepared for each patient who received treatment. The following data were noted upon the card: date, numerical order in EST series for that day, time of administration, time of first awakening (part 1), time of leaving the recovery area (part 2), and special remarks. Notations concerning patient's name, date, serial order, and time of EST administration were made by a nurse at the EST table. The card then accompanied the patient into the recovery area where

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appropriate notations were made by the attendant in charge of that area. He noted the time of first awakening and the time of leaving the recovery area. All time records were maintained accurately to the minute, by synchronized watches.

The recovery procedure utilized for this study was similar to that used before now, since we did not wish to affect results by special departures from our ordinary EST methods. When the patient showed signs of awakening he was called by name and asked whether he wished to go to the breakfast room. Some patients responded at once, dressed and left the room. Others attempted to arise but would then return to sleep for a few minutes to an hour or longer. Occasionally, the patient was startled upon awakening. When spoken to softly he would become calm and relaxed again. Once the patient did leave the recovery area (part 2) he went to the breakfast room where he remained until breakfast was completed. He then had a brief interview with the clinical psychologist. The latter saw the patients in the order in which they had received EST. He interviewed each patient briefly and individually in a small office on the ward, asking these questions:

1. Do you remember hearing any music today?
2. What kind of reaction do you have to it?
3. Is there anything you would like to say about it?
4. How long were you asleep this morning?

When necessary, the interviewer asked additional questions to clarify the patient's response.

Results and Discussion

Although many patients received EST during the period of the experiment, statistical analysis was necessarily limited to small samples for several reasons. Patients were discontinued from treatment at various points during the investigation; some were on maintenance dosage rather than full course of EST. Intercurrent medical illnesses played a role in decreasing sample size. Inspection of the data suggested marked individual difference in speed of recovery. It was therefore necessary to limit most of our analyses to those patients who had gone through all three procedures (Silence, Music A, Music B) in order that results be not altered by inequitable substitutes of subjects among the various methods. For these several reasons, small sample techniques were mandatory.

TABLE I

Speed of Awakening

		(1) Mean Speed of First Awakening	(2) Mean Speed of Leaving Recovery Area	(3) Mean Difference Between (1) & (2)
Silence	(N=7)	37'	87'	50'
Music A	(N=7)	24'	62'	38'
Music B	(N=7)	20'	68'	48'

Table I presents the mean times of recovery under the three different circumstances. Columns 1 and 2 are self explanatory. Column 3 is the difference between these, i.e. the length of time between patient's initial awakening and the time he walks away from the recovery area.

Mean scores for initial awakening time are similar in the case of Music A and Music B, but much longer for Silence. This is also true for time of leaving recovery area (column 2). Time interval between initial awakening and time of leaving recovery area is lowest for Music A, but Music B is similar to Silence.

The significance of these differences in means was statistically evaluated. Table II sets forth the results of t-tests of the significance of the difference between the mean recovery times described in Table I. Music B (bright jazz) definitely leads to more rapid awakening than does Silence ($p < .05$). Furthermore, Music B induces the patients to leave the recovery area more speedily ($p < .001$). Music A shows a trend in this direction, at the .10 level of significance. Each patient was interviewed later in the morning of his treatment and among the questions asked was this: "Do you remember hearing any music today?" Table III reports the number and proportion of patients responding "yes" versus "no" to that question, the first time it was asked following each procedure (Silence, Music A, Music B). Under the assumption that the ratio of "yes" to "no" answers following the condition of Silence reflects the expected extent

TABLE II

Probabilities Drawn From Chi-Square Tests of Differences Among Methods

	Music A (1)	Music A (2)	Music A (3)	Music B (1)	Music B (2)	Music B (3)
Silence (1)	.30	—	—	.05*	—	—
Silence (2)	—	.10	—	—	.001*	—
Silence (3)	—	—	.40	—	—	.70
Music A (1)	—	—	—	.40	—	—
Music A (2)	—	—	—	—	.50	—
Music A (3)	—	—	—	—	—	.40

*Statistically significant difference at the level indicated.

TABLE III

Proportions of Patients Recalling Music After Treatment

	Silence		*A*		*B*	
	No. Patients	Ratio	No. Patients	Ratio	No. Patients	Ratio
Response "No"	13	87%	3	33%	8	73%
Response "Yes"	2	13%	6	67%	3	27%

TABLE IV

Chi-Square Tests of Significance, Yes Versus No

	Chi-Square	*Significance Level*
S* vs B**	13.2***	.001
S* vs A**	1.0	.40
A* vs B**	7.5	.01

*Expected ratio.

**Observed ratio.

***Minimum theoretical frequency raised to 5, thereby reducing Chi-Square to figure reported here.

of departure from the reality situation, Chi-Square tests were made between the obtained versus the expected ratios of "yes" to "no" under each musical condition (Music A and Music B). In each condition the expected ratio of yes:no was 87%:13%. Table IV presents the results of these Chi-Square significance tests between the expected proportions of "yes" and "no" in response to the different methods. Bright jazz (Music B) results in an unusually high number of "no" responses, considerably more so than does quiet classical music ($p < .01$). Its departure from the *expected* yes:no ratio is far beyond that attributable to chance.

It is reasonable to infer that such jazz music (Music B) accentuates confusional effect of EST, insofar as that effect (confusion) is reflected in the misperception of and/or recall of the presence of music. Rank difference correlation coefficients were computed between the patients' subjective estimates of time asleep, and columns 1, 2, and 3 respectively as defined in Table I (time of first awakening, time of leaving recovery area, and difference between these two times.) The only significant correlation obtained was that between subjective estimates of time asleep and time spent in recovery after EST when music was altogether absent (Silence). The data therefore suggest that only during the absence of music, i.e., Silence, is the patient able to make even an approximately valid judgment of the length of time he remained in the recovery area. This reinforces an inference made above, concerning the accentuation of the effect of EST by jazz following the treatment.

In order to test our clinical observation that the individual patient varied considerably in his time of recovery even under the same procedure (Silence, Music A, Music B), reliability coefficients were computed for each procedure. Every patient was compared with himself for his speed of recovery under S, then Music A, and Music B. Only under Music B was there significant consistency between different days for all the measures taken (time of first awakening, time of leaving recovery area, difference between these.) Under the condition of Silence, the difference between first awakening and time of leaving recovery area remained consistent for two different days. The general conclusion here is that bright jazz evokes greater consistency in recovery time for the individual patient. Put differently, it means that an individual patient's speed of recovery may be more readily predicted under bright jazz than under conditions of Silence or of quiet classical music.

A design of the type used for this study was necessary in order that extraneous factors be equalized. For example, the accumulation of fatigue during the week might alter the speed of awakening on Friday after treatment; excitement attendant upon the expectation of visitors or expectation of returning home, emotional reactions to the

previous weekend or effects of visits home, all these could affect results. The primary function of our test of return to consciousness was the measurement of the time of awakening, which was defined as the first obvious indication of visual awareness of external stimuli. The time of rising was also charted as a measurable end point of purposive activity. These times were regarded as measurements of spontaneous change in level of consciousness. More complex criteria were avoided because of the extraneous and perhaps disrupting effect of the test procedures which they entailed.

The initial hypothesis for this study may be answered affirmatively: music during administration of and recovery from EST does affect speed of recovery (awakening). *Type* of music (e.g., bright jazz VS smooth, quiet classical) may be differentially important in this regard. The individual component aspects of music require investigations. What are the differential effects of variations in volume, timbre, pitch? Although music constitutes a configuration which goes beyond the sum of its individual components, it is possible that analytic investigation of its components might yield data such that the effect of an individual musical piece upon speed of post-EST awakening could be predicted.

The findings reported herewith suggest that music potentially can increase the confusional effects of EST. It was noted that bright jazz caused a significantly great proportion of patients to forget and/or to misperceive the presence of music. Furthermore, only in the absence of music was there any relationship between the subjective estimate of sleeping (recovery time) and the objective time of recovery. Should it be verified by other investigations that the confusional effect which follows EST is accentuated by certain qualities of music, then implications ensue concerning the spacing of treatments as in the regressive method which attempts to induce confusion completely and quickly.

It is interesting to note that our bright jazz had those same qualities which Reese,[4] in reviewing the cases of musicogenic epilepsy reported in the world literature, found to be the most dangerous in the music for epileptics: a lively, well punctuated rhythm, instrumental rather than vocal, dance (or marching) music.

Summary

This reports the results of an investigation into the effect of music upon the speed of awakening after EST. Three conditions were created: Silence, Bright Jazz, and Quiet, Classical Music. The design was ABC, BCA, CAB, with three EST treatments weekly for a period of three weeks. Small sample procedures were utilized. Individual interviews were held later in the morning of each treatment.

1. Bright jazz results in greater rapidity of awakening after EST.
2. Few patients recall hearing music when bright jazz is played.
3. When asked to estimate speed of awakening after EST, the patient's judgment is best on the day that music is entirely absent.

A reasonable hypothesis which may be inferred from these data is that bright jazz accentuates the confusion induced by EST. This warrants continued study.

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Book Reviews

PSYCHIATRIC DICTIONARY. Second Edition with Supplement by Leland E. Hinsie, M.D., and Jacob Shatzky, Ph.D., Oxford University Press; 781 p. (1953).

The volume, in its present revision contains over 8000 terms and entries and includes 218 pages more than were contained in the first edition of 1940. The material retains the original format and is liberally interspersed with description and terminology referable to allied fields where the latter may reflect pertinence to psychiatric concepts. A few random references which follow, indicate some illustrative instances of both inclusion and elaboration of terms which, because of the Supplement, have enlarged and revised the book to its present stature as an informative instrument within the framework of psychiatric science.

While *crime* and *intent* are catalogued as such in the original 1940 section, the important forensic problems of relationship between mental disorders and commission of criminal offenses have been elaborately treated under the concepts of *crime and mental disorders, preventives of crime,* and *compulsive-neurotic criminalism.* This new material found in the Supplement, is further discussed from the differential standpoint of biological, psychiatric, and sociological criminology. In addition and for the first time, *criminal* will and *criminal sexual insanity* are also defined. The entry of *traumatophilic diathesis* or accident proneness, a concept of much current psychiatric discussion, is also included in the Supplement. The diagnostic use of the term *psychosomatic* has been elaborated upon, and descriptive reference has been made to the method of research and therapeutic approach as well as to diagnostic affections belonging within or pertaining to this psychiatric concept. The subject of *psychosurgical procedures* has been defined and discussed in some detail, and the presently recognized methods as well as the choice of operative approaches have been outlined under each methodology named. The use of CO_2 *coma* for therapeutic procedures in different types of psychodynamic mechanisms is discussed, and reactions are defined under specific general classes.

The book is authoritative, well documented, and where pertinent references are encyclopedic in format; and the descriptive matter is generously fortified throughout the volume with cross-references and applicable bibliography. Enhanced by its recent supplement it is an up-to-date volume, admirably adapted to use by researchers, authors, practitioners, social workers, psychologists, students, and others either associatively interested or actively participating in the field of psychiatric knowledge.

CURRENT THERAPY—1954. Edited by Howard F. Conn, M.D. Philadelphia: Saunders. Price: $11.00.

The scope and format of the book are excellent, and it has been brought up to date in line with the recent advances in therapy. In the main, the contributors to the volume are of recognized standing in their respective fields and the articles outlining methods of therapy while concisely written, afford ample information in an authoritative, enlightening and practical manner.

While the book is intended for quick reference, its purpose would be markedly enhanced if, coupled with the outlined therapeutic experiences, there would be also uniformly indicated in each headnote with coincident methodology, the contributors' pithy experiential diagnostic description of the condition under consideration. A representative random sampling of descriptive identifications of clinical criteria in the present volume, includes such conditions as Influenza, Viral Pneumonia, Sickle Cell Anemia, Purpuric States, Cirrhosis of the Liver, Cushing's Syndrome, The Allergies, Causalgia, Epilepsy, Headache, Meniere's Syndrome. These are helpful, and the inclusion of such introductory material at the beginning of *each* condition presented, would afford a clearer perspective in understanding the therapeutic problem to follow.

As the roster of pharmaceuticals in the back of the book is incomplete, the list could be made more serviceable by including the absentees, to avoid the need of consulting collateral sources for the information.

The book is well documented, well designed and should be a valuable aid in keeping therapeutically abreast.

Lewis J. Siegal, M.D., LL.B.

BOOKS RECEIVED

PATHOLOGY. By W. A. D. Anderson, M.A., M.D., F.A.C.P., Professor of Pathology and Chairman of Department of Pathology, University of Miami School of Medicine; Director of Pathology Laboratories, Jackson Memorial Hospital, Miami, Florida; Formerly Professor of Pathology, Marquette University School of Medicine, Milwaukee. Second edition. Cloth. Pp. 1393, with 1241 illustrations and 10 color plates. C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, 1953.

THE YEAR BOOK OF NEUROLOGY, PSYCHIATRY AND NEUROSURGERY. (1953-1954 series.) Neurology. Edited by Roland P. Mackay, M.D., Psychiatry. Edited by Nolan D. C. Lewis, M. D., Neurosurgery. Edited by Percival Bailey, M.D., and Oscar Sugar, M.D. Cloth. $6.00. Pp. 604 with 132 illustrations. Year Book Publishers, Inc., 200 E. Illinois Street, Chicago 11, 1954.

APHASIA THERAPEUTICS. By Mary C. Longerich, Ph.D., and Jean Bordeaux, Ph.D. Cloth. $3.75. Pp. 185. The Macmillan Company, 60 Fifth Avenue, New York 11, 1954.